Personality diathesis explains the interrelationships between personality disorder and other mental conditions

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T. Widiger correctly summarizes the often complex relationship between personality disorders and other forms of psychopathology. What is needed now is an integrating hypothesis that both explains existing knowledge and predicts future relationships and developments. I argue here, and have done so previously, that the concept of personality diathesis is such an integrating hypothesis (1). This is a useful concept that combines both latent and overt pathology. Conditions that are best described as diatheses are "mental or physical inherited or acquired chronic predisposition or disease states". For a condition to be described as a diathesis it requires onset early in life, variation in its expression in different settings and at different times, a greater association with other, often more severe, disorders, and its acceptance as an intrinsic component of functioning.

The origin of personality diathesis may be genetic or environmental and subject to often complex interactions between these factors (2), but once it is developed it remains a persistent feature that can be identified. Because it represents a vulnerability, it will have a pathoplastic influence on the presentation of other mental disorders such as depression (3); it will show variation over time, as in the well documented Collaborative Longitudinal Personality Disorders Study, but also show persistence of its core component, inter-personal social dysfunction (4). There may also be a spectrum of disorders within a diathesis – the haemorrhagic diatheses are a good example – but there can be argument over where personality disturbance is on the spectrum. The diathesis model suggests it should be lower down than other disorders but constitutes vulnerability for them.

What is not clear is whether there is more than one diathesis. There are certainly several distinct domains of normal personality, and Widiger and others have argued strongly for the five-factor model to be the integral substrate of DSM-5 personality disorders (5), but there are differences between normal trait expression and the most severe personality disorders (6) that are not merely those of degree. In particular, there is extensive comorbidity between personality disorder categories at greater levels of severity and mental state disorders (6,7) and over a prolonged period there can also be a shift in the expression of personality pathology (8). How these pathologies link together is a matter for further enquiry.

Several conclusions follow from this hypothesis. Firstly, personality disorder presenting later in life is not part of the diathesis. Even though the features may be the same as those with a known disorder (9), the fact that they arise late make them part of a separate diagnosis of "personality change" as described by Widiger. This is an important but poorly studied diagnosis in ICD-10 and a closer look may help in understanding core personality disorders. Secondly, the fail-

ure to diagnose personality disorder in clinical practice, despite its high prevalence in the community (10), is understandable if these conditions are felt to be an intrinsic part of the person rather than a separate disorder or disease. This also explains why approaches that change the environment rather than the person (11) may be valued; they do not alter the diathesis but allow a much better quality of life. Thirdly, the hypothesis leads to the expectation, a word specially chosen as it signifies more than hope, that one or more aspects of personality diathesis can be measured as endophenotypes of disorder, the heritable component of the disorder that is present at all times (12) and which can act as a clinical or biological marker.

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